Pesticide Stressors

Background

The Stanislaus River, between Goodwin Dam and Caswell State Park, has been identified as being impaired on the USEPA Clean Water Act Section 303(d) list for not meeting water quality standards since the early 1990's. The pollutants or stressors that have been identified to cause the impairments are: diazinon, chlorpyriphos, Class A pesticides (e.g., organochlorines), unknown toxicity, mercury, and temperature (USEPA 2011). Some of the beneficial uses that are not being supported include: cold freshwater habitat; migration; spawning, reproduction and/or early development; and warm freshwater habitat. Data evaluated for the most recent update to the 303(d) list confirmed that toxicity to fish species is still a concern in the Stanislaus River (SWRCB 2010), where exposures to river samples resulted in significant reductions in fish survival and growth.

The Central Valley Regional Water Quality Control Board has recently developed a control program and adopted water quality objectives for diazinon and chlorpyriphos in the Central Valley (CVRWQCB 2014), so the implementation of the program should reduce the adverse impacts of these two constituents. However, the use of organophosphate pesticides like diazinon and chlorpyriphos have decline in California since the mid-1990's, and USEPA actions resulted in the phase out of these two pesticides for urban use in the early 2000's (Spurlock and Lee 2008). Much of the pesticide use has shifted to pyrethroids, especially for urban use, and in 2006 pyrethroids accounted for greater than 40% of the insecticide registrations in California. Pyrethroids have been identified as causing much of the surface water and sediment toxicity in California (Anderson *et al.* 2011). More recently, the use of the systemic pesticides neonicotinoids has increased, and their use has been implicated in global declines of some wildlife (Gibbons *et al.* 2014; Mason *et al.* 2012). Current use pesticides are ever changing, and this makes it difficult for regulatory agencies to control the adverse effects that these contaminants create.

The large majority of currently available spawning habitat and subsequent rearing habitat in the Stanislaus River is below Knights Ferry (ESA 2013), and this reach coincides with increased amounts of anthropogenic disturbances, primarily agricultural and urban development. In a review of toxicity monitoring data conducted in California, Anderson and others (2011) found that sites located near agriculture and urban areas had statistically greater occurrences of toxicity in water and sediment samples than near undeveloped areas. In all, 51% and 45% of the streams, rivers, canals, and lakes monitored in 2001-2010 had some toxicity in the water column and sediment, respectively. Toxicological effects can range from sublethal endpoints to full organism mortality. Using correlation analyses and toxicity identification evaluations, Anderson and others (2011) determined that the vast majority of toxicity was cause by pesticides (e.g., insecticides, herbicides, fungicides). However, pesticides were not the cause of all toxicity, and some other contaminants that were identified included metals and ammonia.

Pesticide Toxicity to Fish Species

Fish are not the target organisms of the pesticides; however, pesticides have been found to cause adverse impacts to fish in surface waters. For example, in a review of Central Valley toxicity data, Markiewicz and others (2012) found that the fish species tests, *Pimephales promelas*, had a higher frequency of toxicity than the other species, *Ceriodaphnia dubia* (invertebrate) and *Selenastrum capricornutum* (algal). Samples were toxic to fish in 62% of the tests versus 49% for invertebrates and 40% for algae. Similar to the statewide survey of Anderson and others (2011), pesticides were found to be the primary cause of toxicity in the Central Valley (Markiewicz 2012). Importantly, salmonids generally tend to be more sensitive to chemical stressors than many other species of fish; and, if there are kills of other freshwater fish attributed to use of these pesticides, then it is likely that salmonids have also died (NMFS 2012).

Moreover, the life history strategies salmonids evolved to rely on exposes them to higher risks from contaminants. For example, juvenile salmonids typically occupy and rely on shallow freshwater habitats (e.g., floodplains, off-channel, low flow alcoves) during critical rearing and migratory life history periods. These near-shore, low flow habitats are expected to have higher pesticide loading and concentrations, which subject developing salmonids to higher exposures to pesticides in their preferred habitats (NMFS 2008, 2009, and 2011).

Typically, adult organisms will have a lower risk of morality to contaminants than the sensitive larval fish used for toxicity tests. As a result, toxicity tests with larval fish could overestimate the mortality that might occur to adult salmonids. However, pre-spawn adult salmonids are likely less tolerant to chemical stressors because they have used most of their accumulated fat stores for gamete production (NMFS 2008, 2010, and 2013). It is probable that the some pre-spawn returning adults will die as a result of short-term exposures to pesticides, especially when subjected to additional stressors like elevated temperatures. Additionally, pre-spawn mortality can be cause by other contaminants. For example, metals and petroleum hydrocarbons likely contributed to pre-spawn mortality of coho salmon in urban streams in Washington State (Scholz *et al.* 2011). Pre-spawn mortality is a particularly important factor in the recovery of salmonid populations with low abundance because every adult is crucial to the population's viability (NMFS 2013).

While direct mortality is an obvious detriment to salmonid populations, many sublethal effects of pesticide can also contribute to population declines. Sublethal toxicant exposure often eliminates the performance of fish behaviors, such as predator avoidance, orientation, reproduction, kin recognition, etc. that are essential to fitness and survival in natural ecosystems (Potter and Dare 2003; Scott and Sloman 2004). The most commonly observed links with behavioral disruption include cholinesterase (ChE) inhibition, altered brain neurotransmitter levels, sensory deprivation, and impaired gonadal or thyroid hormone levels (Scott and Sloman 2004). For example, Scholz and others (2000) concluded that olfactory disruption by anti-cholinesterase neurotoxins reduced Chinook salmon anti-predator responses from short-term, sublethal exposures to diazinon. As well, they also concluded that 24-hour exposures to diazinon likely increased the straying of the adult hatchery Chinook salmon over the control group. Furthermore, juvenile salmonids exposed to pesticides during development may fail to imprint to their natal waters, which can lead to increased adulthood straying (NMFS 2009).

Additional evidence of the sublethal effects of pesticides on fish populations have been demonstrated though reproduction experiments. For example, the pyrethroid insecticide cypermethrin inhibited male

Atlantic salmon from detecting and responding to the reproduction priming pheromone prostaglandin, which is released by ovulating females (Moore and Waring 2001). The males exposed to cypermethrin did not respond to prostaglandin with the expected increased levels of plasma sex steroids and expressible milt. In addition, zebrafish exposed to low concentrations (96-hr LC5) of deltamethrin and Achook (a synthetic pyrethroid and a neem based pesticide, respectively) resulted in significant reductions (54% and 18%, respectively) in female fecundity when compared to the controls (Sharma and Ansari 2010). As well, both of the studies found that exposures to pesticides decreased the abundance of hatchlings. The percentage of unhatched fertilized eggs increased in adult zebrafish exposures, and the number of unfertilized eggs increased in salmon egg and milt exposures (Sharma and Ansari 2010; Moore and Waring 2001). Furthermore, the disruption of spawning synchronization could also result in an increase in the number of unfertilized eggs (NMFS 2009).

Herbicide pesticides also have been shown to reduce fish's ability to perform necessary physiological activities. For example, Waring and Moore (1996) observed that concentrations of the herbicide atrazine that showed no lethal effects to Atlantic salmon in freshwater resulted in physiological stress and increased mortality once the fish were exposed to seawater. Subsequent investigations determined that sublethal concentrations of atrazine can reduce $Na^+ K^+$ ATPase activity and the ability of salmon to osmoregulate (Moore and Fewings 2003). Nieves-Puigdoller and others (2007) found similar disruptions in osmoregulation as well as other endocrine disruption, however at higher concentrations of atrazine. Other investigations have concluded that another herbicide, trifluralin, can cause vertebral deformities, which would likely also result in the eventual mortality from predators or reduced prey capture (NMFS 2012). Because pesticides are developed and used for multiple target organisms (e.g., plants, invertebrates, vertebrates), their mechanisms of action are very diverse. This results in a multitude of ways that pesticides can affect salmonid physiology, biochemistry, behavior, etc., and subsequently, many different life stages of salmonids can be adversely impacted.

Indirect Effects of Pesticides

Salmonid populations can also be adversely impacted indirectly by pesticides acting upon their target species. For example, herbicides and insecticides target the food web organisms that the salmonids depend on during rearing and migration. In addition, pesticides in the aquatic environment can shift algal or invertebrate communities to ones that are less nutritious or preferable to salmonids. Modifications to prey and prey food sources can have noticeable effects on fish populations (NMFS 2012). Reduced food for developing salmonids will result in greater competition, reduced fish growth, and possible starvation during critical life stages (NMFS 2008). Other possible indirect impacts to salmonid populations include effects to riparian vegetation (NMFS 2012). Riparian vegetation is important for providing shade, stabilizing stream banks, and providing allochthonous inputs that are important to maintaining salmonid ecosystems.

Population Level Effects

It is very difficult to quantify actual impacts that pesticide stressors have on salmonid populations because the effects can be direct or indirect, lethal or sublethal, long-term or short-term, etc. To determine the possible combined effects that pesticides might have on salmon populations, researchers at the Northwest Fisheries Science Center used models to predict the effects of ChE inhibitors on anadromous Chinook salmon populations in the western United States (Baldwin *et al.* 2009; Macneale *et al.* 2014). The model results indicated that short-term exposures that were representative of real-world seasonal use patterns were enough to reduce the growth and size of juvenile chinook at the time of ocean entry. Consequently, the reduced size at ocean entry was enough to reduce the survival of individuals, which would, over successive years, reduce the intrinsic productivity of the population. Overall, the magnitude of the responses indicates that common pesticides may significantly limit the conservation and recovery of threatened and endangered species in California (Baldwin *et al.* 2009).

Unfortunately, the models only evaluated the direct and indirect effects of the ChE inhibitory pesticides themselves and did not incorporate possible interactions with other types of pesticides, other environmental stressors (e.g., reduced habitat, sub-optimal temperatures), or other contaminants. Different pesticides can work additively to cause a toxic effect, and other contaminants and factors can influence pesticides' effectiveness, as well. For example, through transcriptional assays Hasenbein and others (2014) determined that ammonia likely enhanced the effect of multiple-contaminant exposures to Delta smelt. In addition, copper[1] has also been found to inhibit olfactory responses in salmonids (Hecht *et al.* 2007). Concurrent exposure of salmonids to copper and olfactory inhibitory pesticides can result in toxicological effects, even if both are at concentrations that would not elicit a response in isolation. Furthermore, many pesticides have been found to be able to work synergistically to cause toxicity to salmonids that is multiplicative and not just additive (Laetz *et al.* 2009). Current estimates of the effects of pesticides on salmonids may underestimate the true responses of salmonid populations in surface waters (Baldwin *et al.* 2009).

These additive and synergistic effects from multiple contaminants are true concerns for aquatic environments. For example, in the National Water-Quality Assessment (NAWQA) Program's monitoring of pesticides, they found that more than 90% of the streams located in developed areas contained two or more pesticides or degradates (Gilliom e*t al.* 2006). Furthermore, more than 50% of the streams had five or more pesticides or degradates, and the concentrations of the degradates were often higher than that of the parent pesticide. The degradate forms can be less toxic than the parent pesticide, however, some degradates have been found to be as toxic or more toxic than the parent (Gilliom *et al.* 2006). In addition, pesticide products typically contain additional chemicals like adjuvants, surfactants, solvents, etc. These chemicals are labeled as inert ingredients, but they increase the effectiveness of the active ingredients and can be toxic to non-target species (Beggel *et al.* 2010; Cox and Surgan 2006; Scholz *et al.* 2012). Very little is known about the fate of these "inert" labeled ingredients once they are in surface waters and their possible impacts on salmonid populations.

Pesticide Exposures to Salmonids in the Stanislaus River

Pesticide applications are highly seasonal, and application timing varies by crop type, weather, land use type, etc. Subsequently, pesticide runoff and salmonid exposure to elevated concentrations of pesticides will also be seasonal and affected by other environmental conditions. Quantifying the concentrations of all the pesticides that salmonids are exposed to is difficult. For example, over 1000 pesticide chemicals were applied in California in 2012 (CDPR 2014). In addition, each commodity or crop

[1] Copper is used as an active ingredient in some pesticides; however, pesticides are not the sole source of copper pollution.

type can have multiple pesticide chemicals that are applied to them (e.g., alfalfa crops were associated with greater than 200 pesticide chemicals). Performing chemical analyses, for all possible pesticides in the different reaches of the river where salmonids would be exposed, would not be cost feasible. Furthermore, current analytical methodologies do not allow for all pesticides to be detected at levels that may cause adverse effects to aquatic organisms. For instance, only recently have techniques been developed to reliably detect many pyrethroid pesticides in surface waters at concentrations near or below sensitive species' LC50's (Hladik *et al.* 2009; Mekebri 2011). Even still, LC50 values are concentrations where 50% of the organisms experience mortality. Sublethal effects are likely occurring to salmonid population even if the pesticides or mixtures of pesticides are not detected.

The current limitations of pesticide monitoring in surface waters has prompted the use of models to predict surface water pesticide concentrations and to assess pesticide risks to aquatic organisms. For example, in 2001 the NAWQA program developed a model, Watershed Regressions for Pesticides, to predict atrazine concentrations in national streams (USEPA River Reach File 1; Horn *et al.* 1994), and the program recently expanded the model to predict the concentrations of multiple pesticides (Stone *et al.* 2014). Similarly, the USEPA Office of Pesticide Programs uses various water exposure models to assess the risk of pesticides to aquatic organisms and the environment (USEPA 2014).

Hoogeweg and others (2011) used modeling to quantify the spatial and temporal pesticide risks to threatened, endangered, and other species of concern in the Sacramento River, San Joaquin River, and San Francisco Bay-Delta watersheds. Chinook salmon (Sacramento winter-run, Central Valley spring-run, Central Valley fall-run, and Central Valley late fall-run) and Central Valley steelhead were included on list of nine species of concern. They predicted the frequency that pesticides would exceed aquatic-life benchmarks and the co-occurrence of these exceedances with the species of concern. At least a portion of the Stanislaus River was identified as a "Potential Area of Concern" (i.e., a high frequency of both pesticide exceedances and species richness) in all months except August and November (see Hoogeweg *et al.* 2011, Figures 77-88). However, individual species may still be at risk during these two months because the model does predict that benchmark exceedances would occur, on occasion, during these months.

Summary

Pesticides have a high potential to greatly impact salmonid survival and population recovery. The diverse mechanisms of action of the different types of pesticides found in the aquatic environment have the ability to affect all the life stages of salmonids as well as the ecosystem that they rely on. However, measuring the true impacts of pesticides on salmonid populations is very difficult. As well, the magnitude of pesticide impacts compared to other possible stressors (e.g., temperature, reduced habitat, predation) is unknown. All the stressors likely work in combination to reduce salmonid fitness. Consequently, potential pesticide impacts should be considered with the other stressors for salmonid population recovery, especially in developed areas such as the California Central Valley.

Salmonid Lifecycle Limiting Factors Matrix Evaluation

Hoogeweg and others (2011) modeled of the frequency pesticide exceedances versus the occurrence of all species of concern; however, they did not evaluate risks to individual species, life stages, river reaches, etc. necessary for the limiting factors matrix. Fortunately, the report did include maps of the frequency of aquatic-life benchmark exceedances by month for the entire project area (see Hoogeweg *et al.* 2011, Figures 36-47). To determine the magnitude of pesticide effects on Stanislaus River salmonids, these maps were summarized to determine the relative risk of pesticide exposure by month and river reach (Figure 1 and Table 1). As mentioned earlier, limitations in monitoring and chemical analyses, the multitude of possible pesticide chemicals, etc. precludes the use of strict concentration limitations to evaluate overall pesticide impacts on salmonids throughout the Stanislaus River. In turn, current pesticide impacts to salmonid life stages in the Stanislaus River are based on the relative frequency of pesticides exceeding aquatic-life benchmarks. The target condition for pesticide impacts is zero to little frequency of benchmark exceedances (i.e., Bins 1 & 2 or <5% exceedance).

Tables and Figures

Table 1. Categories of predicted pesticide aquatic-life benchmark exceedances. Frequencies were calculated from the total number of predicted exceedance days for each month for the period of 2000-2009. Any day that had at least one pesticide that exceeded benchmarks was counted as an exceedance day. (adapted from Hoogeweg *et al*. 2011)

Bin Category	Range of the Frequency of Benchmark Exceedances			Severity Ranking
1	0	-	0.017	A
2	0.018	-	0.055	A
3	0.056	-	0.1	B
4	0.101	-	0.153	B
5	0.154	-	0.206	B
6	0.207	-	0.303	B
7	0.304	-	0.447	B
8	0.448	-	0.5	C
9	0.501	-	0.589	C
10	0.59	-	0.994	C

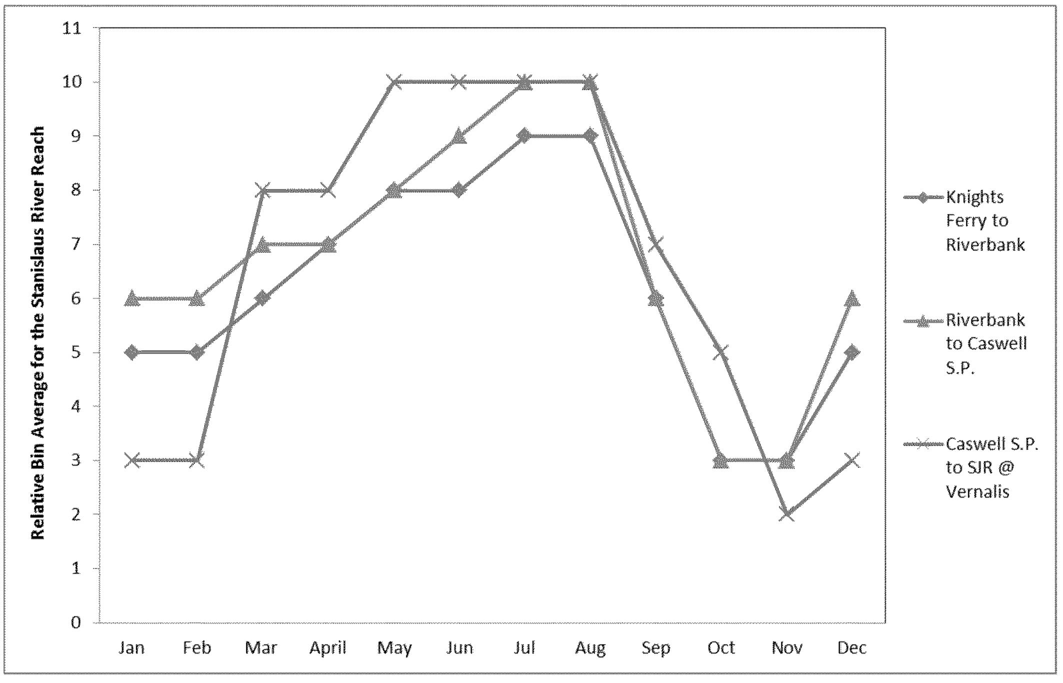

Figure 1. Relative bin value of specified Stanislaus River reaches by month. The values were derived from qualitative averaging of the frequency of benchmark exceedances model maps for years 2000-2009 in Hoogeweg and others (2011). Due to a lack of data, upstream of Knights Ferry in the Stanislaus River was not modeled.

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